

Immunohistochemical Study of Tissue Expression of Trophoblast Cell Surface Antigen 2 (TROP2) Protein in Prostatic Carcinoma and Benign Hyperplasia

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Abstract

Background: Prostate cancer is the second most common malignancy affecting males. TROP2 is a cell surface marker of embryonic stem cells that has been implicated in development of many cancers. **Objective:** This study aimed to investigate the immunohistochemical expression of TROP2 Protein in PCa and BPH tissues and explore TROP2 association with the histopathological parameters of PCa. **Materials and Methods:** a cross-sectional analytical study was conducted in the pathology laboratory of Suez Canal University Hospital on thirty PCa and fifteen BPH specimens (n=45). Sample was collected from the archived blocks of BPH and prostatic carcinoma. TROP2 expression was assessed using immunohistochemistry. **Results:** TROP2 showed positive membranous staining in prostatic epithelial cells of 100% of PCa & in eleven out of fifteen BPH specimens and the difference between the two groups was statistically significant (p-value >0.001). Moreover, high TROP2 expression was found in nineteen PCa and in only four BPH specimens and the difference between the two groups regarding TROP2 staining intensity was also statistically significant (p-value= 0.005). TROP2 showed positive expression in 100% of specimens showing positive perineural invasion and a statistically significant association was found between both TROP2 staining intensity and perineural invasion (p-value >0.05). In addition, TROP2 showed statistically significant validity in discriminating between PCa and BPH (p-value <0.001). **Conclusions:** TROP2 is a sensitive prostatic immunohistochemical marker and its expression in PCa is higher than that in BPH. TROP2 is associated with poor prognostic factors of PCa as perineural invasion.

Keywords: TROP2- Prostatic Carcinoma- Benign Hyperplasia

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Introduction

Prostatic carcinoma is an important health problem of elderly males. As the prostate is a hormone dependent secretory gland, it may undergo a group of diseases namely epithelial hyperplasia, infection, inflammation, benign and malignant neoplastic enlargement.

Most prostatic diseases are benign, however, malignant neoplasms are very important to be studied because of the poor prognosis and high mortality associated with metastases. So, it is important to find new diagnostic tools that allow early tumor detection, better treatment chances and better prognosis [1, 2].

Prostate cancer (PCa) is the second most common malignancy in males next to lung cancer and the fifth cause of cancer death in males worldwide according to WHO in 2020. According to Global Cancer Observatory statistics in December 2020, the Middle East reported 51,649 new prostate cancer diagnoses, corresponding to 3.7% of global cases. In Egypt, PCa was found to be the seventh highly detected cancer representing 7.2% of the total diagnosed cancer cases in Egyptian males [3-5].

Trophoblast cell surface antigen 2 (TROP2), is a stem cell marker that shows membranous expression

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in several normal adult tissues as endocrine, exocrine glands, GIT, heart, kidneys, larynx, salivary gland, skin, thymus, tonsils, trachea, lung, urothelium, prostate, seminal vesicles and female genital tract TROP2 is also a protooncogene whose overexpression is involved in promoting tumor cell growth, proliferation and metastasis [6-8].

TROP2 has been implicated in development and progression of breast and ovarian cancers [9]. TROP2 was found to be important therapeutic target in triple negative breast cancer, advanced lung cancer, and metastatic urothelial carcinoma [6, 10].

Some studies have investigated the expression of TROP2 in prostate cancer and it was found that TROP2 expression was higher in prostate cancer tissue samples compared to normal prostate tissue and that TROP2 expression was associated with poorer overall survival [11-13].

While these studies suggest that TROP2 may play a role in the development and progression of prostate cancer, few studies have investigated the expression of TROP2 in BPH tissue samples [14, 15]. The current study aimed to investigate the immunohistochemical tissue expression of TROP2 proteins in both prostate cancer and BPH tissue samples and explore TROP2 association with the histopathological parameters of PCa to determine if TROP2 protein can be used as potential prognostic biomarkers of prostate cancer. Moreover, this study aimed to give insights into the diagnostic performance of TROP2 expression in differentiating PCa and BPH.

Materials and Methods

The current work is a cross-sectional analytical study, conducted in the pathology laboratory of Suez Canal University Hospital on thirty prostatic carcinoma specimens and fifteen benign prostatic hyperplasia specimens. They were gathered from records and formalin fixed paraffin embedded tissue blocks during the interval between 2013 and 2019.

Hematoxylin & eosin staining

Sections were prepared from the paraffin embedded tissue blocks of PCa and BPH specimens at thickness of 5 μ m and were stained with Hematoxylin & eosin (H&E) stains. H&E slides were initially examined for histopathological features according to the 2022 WHO classification of prostatic tumors that are tumor histologic type, Gleason WHO/ISUP grade grouping, and the estimated approximate percentage of prostate involved by tumor in examined slides, presence or absence of lymphovascular and perineural invasion.

Immunohistochemical staining

Other sections were prepared on positive charged slides to be stained with TROP2 primary antibody. Normal skin tissue was used as a positive control [13]. Immunohistochemical staining for TROP2 was conducted using monoclonal anti-TROP2 IgG clone (F-5): sc-376181 purified antibody that was used according to

the suggested protocol included within companies' data sheets (Santa Cruz).

Staining Technique followed the following steps: at first, sections were deparaffinized in xylene for 30 minutes then rehydrated for 20 minutes in descending grades of alcohol followed by distilled water for 6 minutes. Antigen heat induced epitope retrieval, was done by immersing slides in high pH retrieval buffer solution, for 20 minutes at 98°C in microwave. Slides were cooled gradually followed by immersing in distilled water and wash buffer for 6 minutes. Ten minutes of immersion in hydrogen peroxide was performed for blocking endogenous peroxidase activity followed by immersing in wash buffer for 6 minutes. Slides were incubated with primary antibody for 2 hours at room temperature then washed with wash buffer for 6 minutes before applying the secondary antibody for 25 minutes. After washing with wash buffer the slides were incubated with diaminobenzidine chromogen for 10 minutes, then washed with distilled water then counterstained with ready to use Mayer's hematoxylin for 10 minutes.

Evaluation of immunohistochemical staining of TROP2

PCa and BPH slides stained by TROP2 marker were microscopically examined. The membranous staining of TROP2 in prostatic epithelial cells was quantified based on proportion of stained tumor cells. The membranous expression of TROP2 was scored as negative if percentage of stained tumor cells is less than 10% and as positive if percentage of stained tumor cells is more than 11%. Positive TROP2 expression was subdivided into +1 (low) if percentage of positively stained tumor cells is 11-49% and +2 (high) if percentage of positively stained tumor cells is 50-100% [13].

Data analysis

Descriptive analysis was adopted for percentages and means of different findings. Association between level of TROP2 expression and the different histopathological parameters was calculated using Spearman correlation test and chi square test. Receiver operating characteristic (ROC) curve analysis for TROP2 expression was conducted. Statistical significance was considered at P- value <0.05. Statistical analysis was done using the software SPSS (Version 21) for windows. Visual presentation of results was displayed using tables & figures.

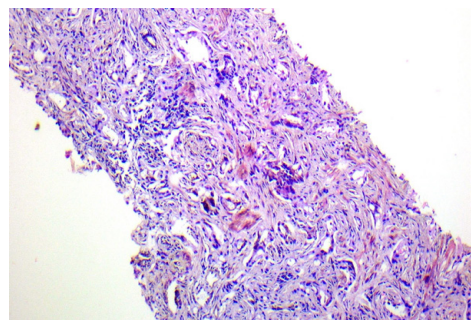


Figure 1. Prostatic Acinar Adenocarcinoma, Gleason grade (4+4), Grade Group 4, H&E Staining, Original Magnification x100.

Table 1. Histopathological Features of PCa Group (n = 30).

Parameters	n
Dominant Growth Pattern	
Acinar	28
Ductal	2
Positive Cribriform Pattern	25
Gleason Grade	
Grade group 3	4
Grade group 4	8
Grade group 5	18
Gleason Score, Median	30
Comedo necrosis	
None	13
Focal	10
Diffuse	7
Approximate % of prostate involved by tumor	
Less than 80%	10
80 – 99%	8
100%	12
Lymphocytic infiltration	12
Positive LVI	5
Positive PNI	9

LVI, Lymphovascular Invasion. PNI, Perineural Invasion

Results

A total of forty five prostatic specimens were included in the present study, of which thirty were PCAs (Table 1) (Figure 1) and fifteen were BPH (Figure 2).

TROP2 immunohistochemical staining in benign prostatic hyperplasia and prostatic carcinoma specimens

TROP2 expression showed membranous staining in in prostate basal epithelial cells of 100% (thirty) of PCa (Figure 3 and 4) and in eleven BPH specimens. TROP2 expression was negative in four BPH specimens (Figure 5) with statistically significant difference between the two groups (p-value= 0.009) (Table 2).

As regards TROP2 staining intensity, high TROP2 expression was found in nineteen PCa (Figure 3) and in only four BPH specimens and low TROP2 expression was found in seven BPH and in only eleven PCa specimens (Figure 4) and the difference between the two groups concerning staining intensity was statistically significant (p-value= 0.005) (Table 2).

Association between TROP2 immunohistochemical staining and histopathological characteristics in prostatic carcinoma group

Regarding Gleason grade, among nineteen specimens that showed high TROP2 expression, eleven specimens were Gleason grade group 5, five specimens were Gleason grade group 4 and only three specimens were Gleason grade group 3. TROP2 expression was also high in eight out of twelve specimens showing positive lymphocytic

infiltration. Low TROP2 expression was found in three out of five specimens revealing positive lymphovascular invasion. However, p-value of association between TROP2 expression and all these studied histopathological parameters of PCa was greater than 0.05. Interestingly, a statistically significant association was found between TROP2 staining intensity and perineural invasion (p-value >0.05) (Table 3).

Receiver operating characteristic (ROC) curve analysis for TROP2 expression

Regarding the diagnostic performance of TROP2 expression in differentiating PCa and BPH, TROP2 showed statistically significant validity in discriminating between PCa and BPH (p-value <0.001) (Table 4, Figure 6). TROP2 showed 100% sensitivity and 73.33% specificity in detecting PCa (Table 5).

Discussion

The study was performed on thirty prostatic carcinoma specimens and fifteen benign prostatic hyperplasia specimens. TROP2 showed positive membranous expression in all prostatic carcinoma and eleven out of fifteen BPH specimens and the difference between the two groups was statistically significant (p-value >0.001).

The findings are largely comparable to those found in other research that studied TROP2 expression in prostatic tumors, in which TROP2 revealed positive membranous expression in prostatic epithelial cells and its expression in PCa cells ranged between 63.4 & 100% & was higher

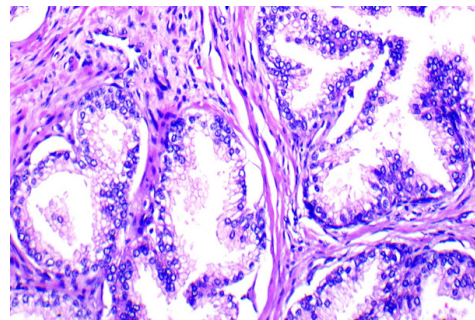


Figure 2. Benign Prostatic Hyperplasia Showing Dilated Prostatic Acini with Double Layered Epithelium, H&E Staining, Original Magnification x400.

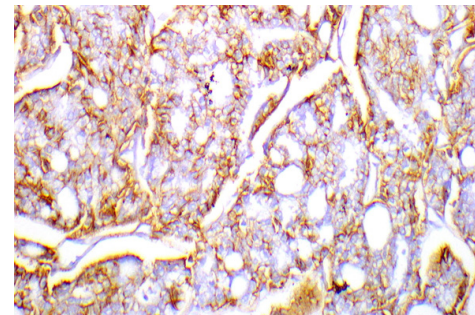


Figure 3. Positive TROP2 Membranous Staining Score 2 in Prostatic Acinar Adenocarcinoma, Gleason Grade (4 +4), Grade Group 4, Immunohistochemical Staining, Original Magnification x200.

Table 2. TROP2 Immunohistochemical Staining in PCa and BPH Specimens

Parameters	BPH (n = 15)	PC (n = 30)	p-value
TROP2 Expression			
Negative	4 (26.7%)	0	0.009 *F
Positive	11 (73.3%)	30 (100.0%)	
Median percentage of TROP2 positivity			
Median	20.0 (0.0 – 55.0)	85.0 (40.0 – 90.0)	<0.001 *M
TROP2 Staining Intensity			
No Staining	4 (26.7%)	0	0.005 *F
Low	7 (46.7%)	11 (36.7%)	
High	4 (26.7%)	19 (63.3%)	

BPH: Benign Prostatic Hyperplasia, PCa: Prostatic Carcinoma, TROP2: Trophoblast Cell Surface Antigen 2, *statistically significant p-value at $p < 0.05$, F. Fischer's exact test, M. Mann Whitney test.

Table 3. Association between TROP2 Expression and Histopathological Parameters in PCa Group (n = 30).

Parameters	n	TROP2 expression		p-value
		Low (n=11)	High (n=19)	
Dominant Growth Pattern				
Acinar	28	10 (90.9%)	18 (94.7%)	1.00 F
Ductal	2	1 (9.1%)	1 (5.3%)	
Positive Cribriform Pattern	25	10 (90.9%)	15 (78.9%)	0.626 F
Gleason Grade				
Grade group 3	4	1 (9.1%)	3 (15.8%)	1.00 F
Grade group 4	8	3 (27.3%)	5 (26.3%)	
Grade group 5	18	7 (63.6%)	11 (57.9%)	
Gleason Score, Median	30	9.0 (8.0 – 9.0)	9.0 (8.0 – 9.0)	0.800 M
Comedo necrosis				
None	13	4 (36.4%)	9 (47.4%)	0.622 F
Focal	10	5 (45.5%)	5 (26.3%)	
Diffuse	7	2 (18.2%)	5 (26.3%)	
Approximate % of prostate involved by tumor				
Less than 80%	10	3 (27.3%)	7 (36.8%)	0.095 F
80 – 99%	8	1 (9.1%)	7 (36.8%)	
100%	12	7 (63.6%)	5 (26.3%)	
Lymphocytic infiltration				
Positive LVI	5	3 (27.3%)	2 (10.5%)	0.327 F
Positive PNI	9	6 (54.5%)	3 (15.8%)	0.042 *F

TROP2: Trophoblast Cell Surface Antigen 2, M. Mann Whitney test, F. Fischer's exact test. LVI: Lymphovascular Invasion. PNI: Perineural Invasion. * Statistically significant p-value at $p < 0.05$.

than its expression in benign prostatic epithelial cells (75%) [13-15]. This variation in percentage of expression between studies is mostly related to differences in sample size, type of specimens & primary antibody nature.

Additionally, TROP2 staining differed between PCa and BPH groups in its intensity (p -value = 0.005), in which high TROP2 expression was found in nineteen PCa and in only four BPH specimens and low TROP2 expression was found in seven BPH and eleven PCa specimens. Comparable finding was reported in previous study [14] which also documented that high TROP2 intensity was more in PCa (50%) than in BPH specimens (30%) and low TROP2 intensity was also greater in BPH study group (45%).

Our study observed a positive association between TROP2 intensity and Gleason grades, where specimens with higher Gleason grades exhibit stronger TROP2 staining. This correlation highlights TROP2's role in prostate cancer aggressiveness, supporting its known function in enhancing the expression of proto-oncogenes and promoting self-renewal and proliferation of cancer cells. TROP2 upregulation may thus contribute to the progression and malignant behavior of prostate cancer, with more aggressive cases showing stronger TROP2 expression [8, 14, 16, 17].

However, despite the observed trend, association between TROP2 intensity and Gleason grade wasn't statistically significant. This finding is in line with several

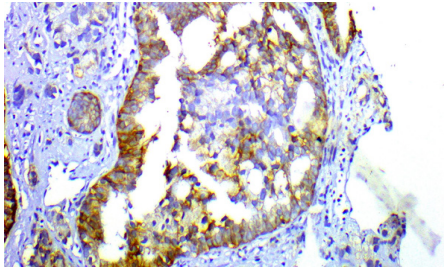


Figure 4. Positive TROP2 Membranous Staining Score 1 in Prostatic Acinar Adenocarcinoma, Grade (4 +3), Grade Group 3, Immunohistochemical Staining, Original Magnification x200

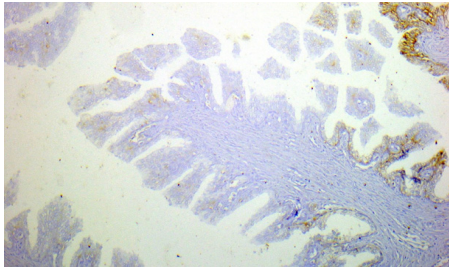


Figure 5. Negative TROP2 Membranous Staining Score 0 (less than 10%) in Benign Prostatic Hyperplasia, Immunohistochemical Staining, Original Magnification x100

studies, though it contrasts with research by Akarken & Dere (2021), which reported a statistically significant link between TROP2 expression and Gleason grades. Such discrepancies might arise from variations in sample sizes, TROP2 scoring methodologies or interpretation of staining intensity across studies.

The present work showed positive TROP2 was in all (nine) specimens with positive perineural invasion, with a statistically significant association (p -value >0.05). This result aligns with previous findings by Akarken & Dere (2021) and partially reflects TROP2 involvement in promoting PCa invasiveness. TROP2 is thought to contribute to perineural invasion by facilitating epithelial-mesenchymal transition (EMT) through the upregulation of p-21-activated kinase 4 [8, 17].

Moreover, TROP2 was expressed in all (five) prostatic carcinoma samples that showed positive lymphovascular invasion. Unlike perineural invasion, however, this association was not statistically significant in our study,

which diverges from Akarken & Dere’s (2021) findings of a significant association between TROP2 expression and lymphovascular invasion. This discrepancy may stem from the small sample size of specimens with lymphovascular invasion in our research or differences in statistical methods. Nonetheless, the consistent expression of TROP2 in specimens with invasive features suggests its potential importance in PCa progression and highlights the need for further investigation into its role in lymphovascular invasion and other aggressive cancer behaviors.

When studying the diagnostic performance of TROP2 in discrimination between PCa and BPH, we found that TROP2 exhibited a sensitivity of 100%, which indicates its strong ability to detect malignant prostatic lesions. However, TROP2’s specificity was only 73.33%. The positive predictive value (PPV) and negative predictive value (NPV) for TROP2 were 88.2% and 100% respectively.

TROP2 demonstrated statistically significant validity in differentiating Prostatic Carcinoma from Benign prostatic hyperplasia (p -value < 0.001). The diagnostic strength of TROP2’s sensitivity offers a promising strategy for accurately identifying malignant prostatic lesions. This study is the first to document the diagnostic capability of TROP2 in distinguishing PCa from BPH suggesting that this marker could improve early detection, diagnosis, and prognosis of prostatic carcinoma in clinical practice.

In conclusion, TROP2 is a sensitive prostatic immunohistochemical marker and its expression in PCa is higher than that in BPH. TROP2 is associated with poor prognostic factors of PCa as perineural invasion.

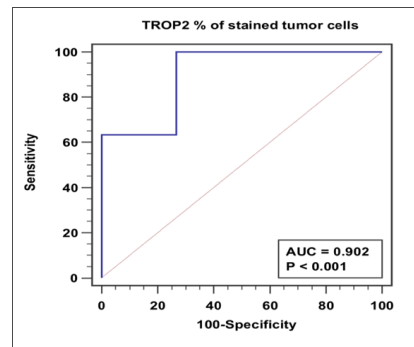


Figure 6. ROC Curve of TROP2 Expression Level for Discrimination between PCa and BPH (n = 45).

Table 4. Diagnostic Performance of NPM1 and TROP2 Expression Levels for Discrimination between PCa and BPH (n = 45).

	AUC	Standard Error	95% CI	z statistic	p-value (Area=0.5)
TROP2	0.902	0.0495	0.776 to 0.970	8.13	$<0.001^*$

AUC: Area under the ROC curve. CI: Confidence interval

Table 5. Cut-off Values of NPM1 and TROP2 Expression Levels for Discrimination between PCa and BPH (n = 45).

	Cut-off value	Sensitivity	Specificity	+LR	-LR	+PV	-PV
		95% CI	95% CI	95% CI	95% CI	95% CI	95% CI
TROP2	>25	100 (88.4-100)	73.33 (44.9 -92.2)	3.8 (1.62 -8.68)	0	88.2 (76.4-94.6)	100

CI: Confidence interval. LR: Likelihood ratio. PV: Predictive value

So, addition of TROP2 to the immunohistochemical diagnostic panel of prostatic carcinoma or targeting it in therapy regimens could improve outcome of prostatic carcinoma patients.

It is recommended to further evaluate TROP2 expression in large scale studies with bigger sample size including different histological subtypes of prostatic carcinoma together with follow up of prostatic carcinoma specimens that shows significant TROP2 expression to monitor survival rates and prognosis of patients. This may provide insight into prognostic significance of TROP2 expression in prostatic carcinoma.

Declarations

Availability of data and materials

All data generated or analysed during this study are included in this article.

Competing interests

The authors declare that they have no competing relevant financial or non-financial interests to disclose.

Author contributions

M.A., M.H., M.O., S.F., M.K. contributed to research conception and design, data acquisition, statistical analysis, data analysis, interpretation, drafting and critical revision of the manuscript. M.H., M.O., S.F., M.K. commented on previous versions of the manuscript. M.A., M.H., M.O., S.F., M.K. read and approved the final manuscript.

Ethical approval

The study protocol was reviewed and accepted by the institutional review board and institutional research ethics committee of Faculty of Medicine Suez Canal University prior to initiation (22/3/2023), s.no (5306).

The research involved no risks over researcher, sample or environment.

The need for informed consent has been waived by the institutional review board and institutional research ethics committee of Faculty of Medicine Suez Canal University.

The study used human data and material (paraffin blocks) and it was adherent to the declaration of Helsinki and this was insured by the institutional review board and institutional research ethics committee of Faculty of Medicine Suez Canal University.

References

1. Rebello RJ, Oing C, Knudsen KE, Loeb S, Johnson DC, Reiter RE, Gillissen S, Van der Kwast T, Bristow RG. Prostate cancer. *Nature Reviews. Disease Primers*. 2021 02 04;7(1):9. <https://doi.org/10.1038/s41572-020-00243-0>
2. Peyromaure M, Ravery V, Boccon-Gibod L. *Prostate Cancer. Textb Men's Heal Aging* 2. 2023;131–46.
3. Kearney G, Chen M, Mula-Hussain L, Skelton M, Eren MF, Orio PF, Nguyen PL, D'Amico AV, Sayan M. Burden of prostate cancer in the Middle East: A comparative analysis based on global cancer observatory data. *Cancer Medicine*. 2023 Dec;12(23):21419-21425. <https://doi.org/10.1002/cam4.6689>
4. Zhang W, Cao G, Wu F, Wang Y, Liu Z, Hu H, Xu K. Global Burden of Prostate Cancer and Association with Socioeconomic Status, 1990-2019: A Systematic Analysis from the Global Burden of Disease Study. *Journal of Epidemiology and Global Health*. 2023 09;13(3):407-421. <https://doi.org/10.1007/s44197-023-00103-6>
5. Elshnoudy IA, Elkhoully AM, Masoud M, Rabea HA, Mansour FR. Medicinal plants cultivated in Egypt with anticancer potential; a systematic review. *Phytochem Rev* 2024. 2024;1–57.
6. Shvartsur A, Bonavida B. Trop2 and its overexpression in cancers: regulation and clinical/therapeutic implications. *Genes & Cancer*. 2015 03;6(3-4):84-105. <https://doi.org/10.18632/genesandcancer.40>
7. Lenárt S, Lenárt P, Šmarda J, Remšík J, Souček K, Beneš P. Trop2: Jack of All Trades, Master of None. *Cancers*. 2020 Nov 11;12(11):3328. <https://doi.org/10.3390/cancers12113328>
8. Qiu S, Zhang J, Wang Z, Lan H, Hou J, Zhang N, Wang X, Lu H. Targeting Trop-2 in cancer: Recent research progress and clinical application. *Biochimica Et Biophysica Acta. Reviews on Cancer*. 2023 07;1878(4):188902. <https://doi.org/10.1016/j.bbcan.2023.188902>
9. Chen R, Lu M, Wang J, Zhang D, Lin H, Zhu H, Zhang W, Xiong L, Ma J, Mao Y, Zhu J, Xu J. Increased expression of Trop2 correlates with poor survival in extranodal NK/T cell lymphoma, nasal type. *Virchows Archiv: An International Journal of Pathology*. 2013 Nov;463(5):713-719. <https://doi.org/10.1007/s00428-013-1475-4>
10. Zeng P, Chen M, Zhou L, Tang M, Liu C, Lu P. Impact of TROP2 expression on prognosis in solid tumors: A Systematic Review and Meta-analysis. *Scientific Reports*. 2016 09 20;6:33658. <https://doi.org/10.1038/srep33658>
11. Stoyanova T, Goldstein AS, Cai H, Drake JM, Huang J, Witte ON. Regulated proteolysis of Trop2 drives epithelial hyperplasia and stem cell self-renewal via β -catenin signaling. *Genes & Development*. 2012 Oct 15;26(20):2271-2285. <https://doi.org/10.1101/gad.196451.112>
12. Garraway IP, Sun W, Tran CP, Perner S, Zhang B, Goldstein AS, Hahm SA, et al. Human prostate sphere-forming cells represent a subset of basal epithelial cells capable of glandular regeneration in vivo. *The Prostate*. 2010 04 01;70(5):491-501. <https://doi.org/10.1002/pros.21083>
13. Trerotola M, Ganguly KK, Fazli L, Fedele C, Lu H, Dutta A, Liu Q, et al. Trop-2 is up-regulated in invasive prostate cancer and displaces FAK from focal contacts. *Oncotarget*. 2015 06 10;6(16):14318-14328. <https://doi.org/10.18632/oncotarget.3960>
14. Hsu E, Rice MA, Bermudez A, Marques FJG, Aslan M, Liu S, Ghoochani A, et al. Trop2 is a driver of metastatic prostate cancer with neuroendocrine phenotype via PARP1. *Proceedings of the National Academy of Sciences of the United States of America*. 2020 01 28;117(4):2032-2042.

<https://doi.org/10.1073/pnas.1905384117>

15. Akarken İ, Dere Y. Could trop-2 overexpression indicate tumor aggressiveness among prostatic adenocarcinomas?. *Annals of Diagnostic Pathology*. 2021 02;50:151680. <https://doi.org/10.1016/j.anndiagpath.2020.151680>
16. Shen M, Liu S, Stoyanova T. The role of Trop2 in prostate cancer: an oncogene, biomarker, and therapeutic target. *American Journal of Clinical and Experimental Urology*. 2021;9(1):73-87.
17. Lombardi P, Filetti M, Falcone R, Altamura V, Paroni Sterbini F, Bria E, Fabi A, et al. Overview of Trop-2 in Cancer: From Pre-Clinical Studies to Future Directions in Clinical Settings. *Cancers*. 2023 03 13;15(6):1744. <https://doi.org/10.3390/cancers15061744>



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